Atherosclerosis of radial arterial graft may increase the potential of vessel spasm in coronary bypass surgery

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Radial artery (RA) grafts are increasingly used as the second arterial conduit in coronary artery bypass grafting procedures because of recent studies demonstrating favorable early and midterm clinical results.[1] However, the current literature does not provide clear guidelines regarding the appropriateness of using RA with atherosclerotic changes, a situation frequently observed intraoperatively. We compared the in vitro electrophysiologic properties of atherosclerotic and normal RA smooth muscle cells in an effort to characterize the physiologic changes of atherosclerosis.

Materials and Methods

RA samples were collected during the procedure (with preoperative patient consent) and shipped to the research laboratory in 0.9% saline solution on ice. Intraoperative visual inspection was used to determine the presence or absence of atherosclerotic plaques, which was then confirmed by light microscopic examination of the endothelium in the laboratory. Photographs of normal and sclerotic vessels were taken with a digital camera with a 1:2 macro lens. The arteries were immediately transferred to oxygenated Krebs solution (4°C), and the connective tissue was removed. The smooth muscle strip was carefully dissected and dissociated in an enzyme tube containing collagenase (type F, 1 mg/mL) and elastase (type IV, 0.12 mg/mL). The tissues were incubated with the enzyme in a 37°C bath for 45 minutes and carefully triturated to liberate free smooth muscle cells.

Electrophysiologic responses were tested in cells that were phase dense and appeared relaxed. Cell membrane potential was recorded at room temperature with current-clamp mode after a gigaohm seal was obtained. Potassium and calcium currents were also examined with voltage-clamp for kinetic and pharmacologic characterization (data not presented). Acquisition and analysis of data were accomplished with Axopatch 200B and pCLAMP8 software (Axon Instruments, Inc, Foster City, Calif).

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Received for publication Nov 5, 2004; accepted for publication July 19, 2005.

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J Thorac Cardiovasc Surg 2005;130:1477-8

0022-5223/$30.00

doi:10.1016/j.jtcvs.2005.07.048

Results

Three atherosclerotic arteries were found among 38 RA samples in the study. These arteries were obtained from patients with no contraindications to coronary artery bypass grafting. Fat plaques and loss of elasticity of arterial wall were used as the diagnostic criteria. The membrane potentials of normal RA range from −30 to −70 mV, compared with −10 to −35 mV in their sclerotic counterparts. The average membrane potentials of normal and atherosclerotic RA tissues were -50.0 ± 7.8 mV ($n = 4$) and -20.3 ± 6.0 mV ($n = 3$, $P < .05$), respectively. One of the sclerotic tissues also exhibited spontaneous spikelike hyperpolarizations (indicative of sporadic activation of calcium-dependent potassium ion currents); this type of activity was not seen in any of the normal tissues. The membrane potential recordings of both groups are shown in Figure 1.

Discussion

Arterial conduits—internal thoracic artery, gastroepiploic artery, and RA—are generally favored relative to saphenous vein grafts because of superior long-term graft patency and patient survival.[2] The RA graft in particular, with its easy handling characteristics, versatility, and availability, is quickly becoming the second conduit of choice after the left internal thoracic artery.

Various degrees of atherosclerosis are frequently observed intraoperatively in RA. It is estimated that 5% of RAs have atherosclerotic changes,[3] although we did notice a higher incidence in our study. The physiologic properties of RA grafts have not been extensively studied,[4] and even less is known about atherosclerotic RA. Without any scientific evidence, the decision to accept or reject a graft has largely been arbitrary. Such a decision can be particularly problematic in situations where the choices of alternative conduits are limited, such as in patients with severe varicosities or in reoperations.

The resting membrane potential of vascular smooth muscle is mainly determined by transmembrane potassium efflux. The elevated membrane potentials and spontaneous activity seen in sclerotic tissues, but not in normal tissues, suggest calcium ion handling may be compromised, leading to excessive calcium-sparking activity and activation of a variety of ionic currents. The end result of these changes is increased excitability and vasoconstriction.

RA has a relatively thick smooth muscle layer relative to ITA and is more likely to spasm. It is well documented that spasm and stringing comprise the major mode of early RA graft failures. With the higher resting membrane potential of atherosclerotic RA, it is likely that it is even more susceptible to graft spasm. We therefore caution against the use of RA with atherosclerosis unless otherwise unavoidable, in which case maximum pharmacologic antispasmodic treatments should be implemented. More vigilant postoperative monitoring and aggressive investigations are also warranted in these patients when spasm is suspected.

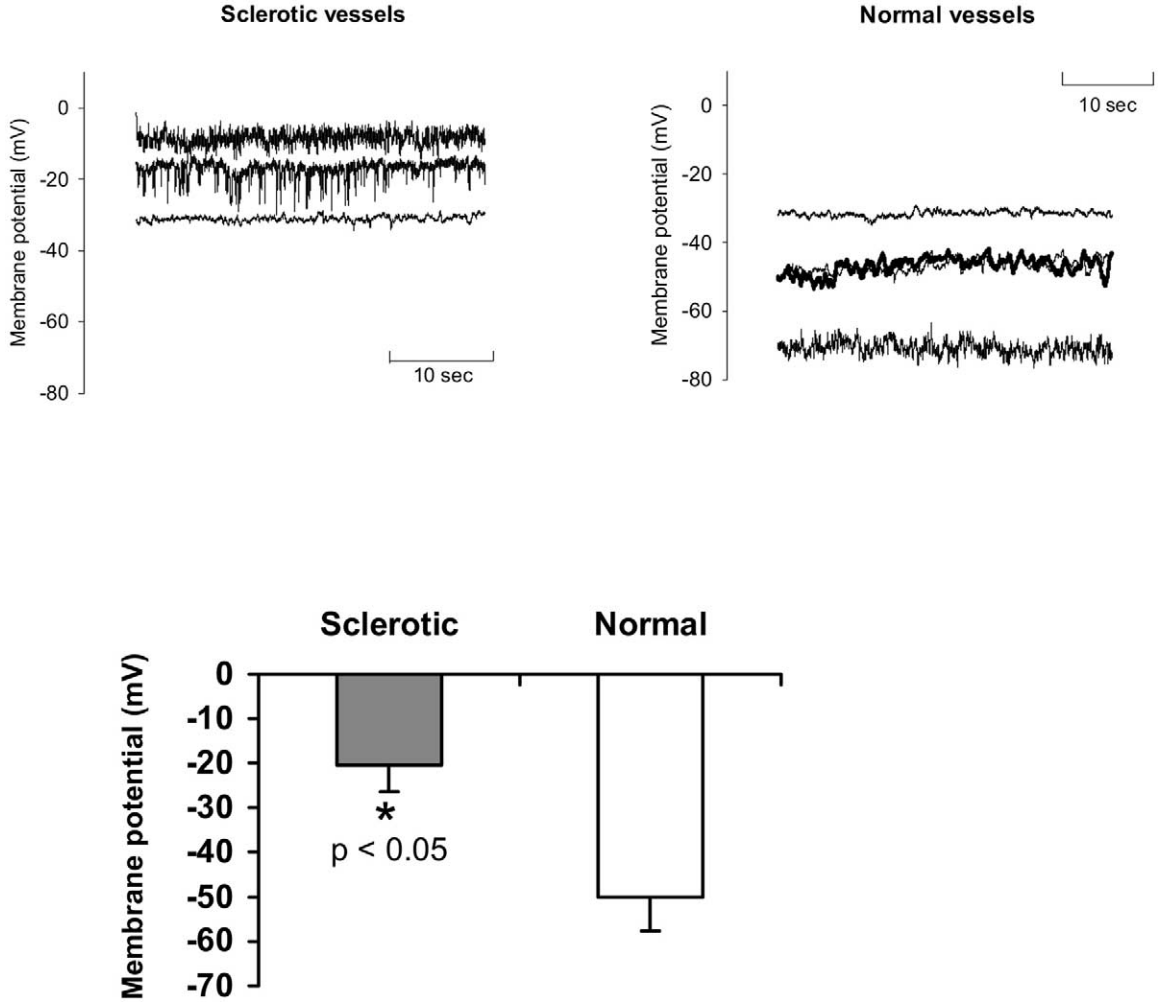

Figure 1. Membrane potential recordings of sclerotic and normal RAs.

Limited by the scope of our study, we did not conduct pathologic grading of RA atherosclerosis. Future studies correlating the degree of membrane potential impairment and the severity of the atherosclerosis are planned.

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